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PSYCHIATRY AND MEDICINE*

By Karl A. Menninger, M.D.

To obtain a proper orientation as to the relationship of psychiatry to the general practice of medicine, one must consider its historical development. For our purposes it is unnecessary to go back further than the Middle Ages, since psychiatry in its present day form had its incipiency in that era. Medicine, on the other hand, was already an ancient discipline and its development in the Middle Ages was coordinate with religion, and with the crafts. I select these three phases of human activity because from them developed what is now psychiatry.

Medicine, at that time, was largely a *chemical* discipline, dependent upon alimentary therapy in both theory and practice. This it had inherited from antiquity. But from the practical guild- and craftsmen there developed various *physical* and mechanical devices applicable to medical practice and these were gradually assimilated by Mother Medicine. From midwifery (a craft) came obstetrics; from tooth-pulling came dentistry; from barbering came surgery. What we now call neurology was always an integral part of medicine, an elaborate, interesting but unproductive part. The marriage of practical surgery with respectable neurology resulted in a very lusty child—neurosurgery—which revived neurology and made it useful as well as respectable.

Meanwhile, from the vague assemblage of ideas represented by religion, philosophy, alchemy and astrology, there developed a descriptive interest in certain unclassifiable, unpredictable, irregular psychological phenomena characterized chiefly by social incompatibility. The persons exhibiting this behavior were called criminals, witches, the bewitched, or the insane, and these names gradually became established as *legal* designations. From among them, finally, the most unpredictable, incomprehensible, and paradoxically behaving individuals were singled out for political detention and gradually fell into the hands of scientists, who studied the behavior and found it to be less incomprehensible than had been assumed, indeed entirely under-

* An address delivered before the St. Louis County Medical Society, May 5, 1936 in conjunction with addresses by Dr. Adolf Meyer and Dr. Abraham Myerson.

standable once all the facts became known. These scientists were, although working with persons acknowledged to be sick, haltingly accepted by Mother Medicine, at first, but with the unexpected demands of the World War, psychiatry became the Cinderella specialty and advanced to great favor. The most important result of this was that the psychological component in the human personality was recognized along with the chemical, mechanical, and physical derivatives.

The sudden great popularity of psychiatry almost ruined it because the great promise held out by the recognition of this previously ignored element in disease was not fulfilled. Too much was claimed by, and too much was expected of the new science. As had been so often said of neurology, psychiatry could offer diagnosis but not cure. This was vividly true in the cases diagnosed as psychopathic personality, psychoneurosis, and schizophrenia.

Fortunately, in the nick of time, psychiatry, like neurology, took unto itself a fertile bride and its marriage to psycho-analysis had the same rejuvenating and fructifying effect as did the union of neurology and surgery. This does not mean, of course, that all psychiatric therapy is psychoanalytic, any more than all neurologic therapy is surgery. But the advent of psychoanalysis ended the era of therapeutic nihilism in psychiatry.

From exclusive application to those patients with obvious and extreme psychopathology, psychiatry was gradually extended to other fields—criminology, sociology, pedagogy, and recently, with increasing intensity, to the parent field, general medicine. To it psychiatry brought techniques of approach to the patient, of personality evaluation and of therapeutic management with which general medicine for all its perfection of laboratory procedure and clinical measurement had been only vaguely familiar. Here and there an intuitive old general practitioner had discovered for himself in the "art" of medicine the importance of dealing with the *feelings* of the patient and of influencing his recovery by a proper consideration of these intangibles. But psychiatry, with the aid of psychoanalysis, has made progress in the reduction of this intuitive technique to systematic, scientific principles, the application of which need no longer be limited to a gifted few.

As a result, the emphasis on *psychological* factors in disease (and in health) is slowly permeating the entire practice of medicine. No longer the exclusively *chemical* concepts of the Middle Ages and earlier,

nor the chemico-*physical* concepts of the nineteenth century, but a concept of interacting physical *and* chemical *and* psychological factors begins, now, to characterize the theory and practice of medicine. Once it was considered ridiculous to record a temperature; later it was considered ridiculous to examine a specimen of urine; and when these procedures became generally accepted it was still considered ridiculous to inquire into the details of a patient's dream. Now we know that dreams and urine and fever may and *must* all be examined if we are to truly and fully understand a patient. No intelligent or informed physician thinks any of these procedures absurd today—even though in practice he may sometimes omit one or several of them. For every thoughtful physician knows that psychological factors are as real and as effective as physical or chemical factors. His training, however, makes him feel more proficient and sure of himself in the old traditional modalities of physics and chemistry and he is often afraid to venture into the psychological investigations that he fully realizes his patients merit and need.

The result is that certain rather sharp differences persist in the points of view and techniques of most medical men, on the one hand, and psychiatrists on the other. The psychiatrists sometimes feel that they alone actually look at the human being as a whole, instead of as a creature with certain extraneous pathological attachments or invasions. The general medical man, on the other hand, suspects the psychiatrist of ignoring or underestimating physical and chemical data and of exaggerating psychic factors beyond their proper degree of importance. He thinks, too, that psychiatrists are apt to see the whole world a little askew, as if all people were "more or less crazy" and this strikes him as ill-founded and distorted.

Then, too, the time concepts in psychiatry and in most other medical specialties (phthisiology and orthopedics excepted) differ considerably. Psychiatry, along with the two specialties mentioned, thinks in long-time terms; treatment extends over a long period, prognosis considers a long interval; the patient's whole life is under consideration rather than an acute episode or relatively brief illness.

Again, the patients with whom psychiatrists deal are often under some degree of social taboo which does not apply to the patients of most other specialties, that of the urologists perhaps excepted.

Finally, the psychiatric modalities depend more upon aural than

upon oral ministrations. Words, i.e., ideas are "poured into" the patient rather than drugs, with, however, no less palpable—often identical—effects. This is a difference which the general medical man accepts with grave misgivings, because he feels suspicious of anything which resembles, even remotely, the unsavory procedures of quackery and faith healing. Unfamiliar with the deeper mechanisms of psychological reactions, he cannot understand the therapeutic results which shrewd cult leaders often secure, but has a strong feeling of condemnation for them because of their unscientific and often obviously dishonest methods. Because psychiatrists work with the same raw material and must use scientifically some of the tools which have fallen into the hands of unscrupulous and unreliable persons in some instances, the reproach attached to the faith healers has often been turned against them unjustly.

But the essential differences that yet separate psychiatry from general medicine go deeper than this. They may best be illustrated by indicating the points of routine psychiatric inquiry and comparing these with general medical practice.

To the psychiatrist, how the patient *acts and reacts* (behaves and feels) are important data. To the average physician, they are not. To the patient, they may or may not be.

For example, a cardiac patient may describe in detail his subjective reactions during a moving picture show or during an attack of decompensation; in either case the average physician listens only from courtesy, if at all. The psychiatrist, on the other hand, puts great stock in such data. The patient may or may not see any sense in so doing.

Similarly, the psychiatrist is just as interested in how a patient reacts to the making of a physical examination as he is in the other data obtained from such a physical examination. He is just as interested in observing what the patient's emotional reactions are toward his office nurse or the hospital manager or himself as he is in discovering what the patient's blood pressure or leucocyte count or vital capacity may be.

The psychiatrist considers it the first task of a physician to observe these ordinarily neglected data, to recognize them, to appreciate that they have meaning, to seek this meaning, to try to evaluate them. He feels that only having done this can he decide what should be done next

with any degree of scientific precision. This "next" may be a chemical manipulation, a mechanical adjustment, or psychological therapy.

It is, of course, impossible to definitely separate diagnosis, prognosis, and treatment; and the psychiatrist feels that attention to these data of action and reaction are not only necessary for a diagnosis and for the prescription of treatment but he feels that often the first indicated treatment is to let the patient know that he, the doctor, is actually interested in these data; he also feels that the prognosis can only be accurately determined by considering them.

For that reason the psychiatrist routinely investigates elaborately the historical data connected with the development of the personality as well as of the present illness. At the same time he does *not* neglect (as he is sometimes thought to) an exhaustive physical inventory, including the routine physical, laboratory, and x-ray investigations, specialized neurological examinations as well, and, finally, a very careful, systematic psychological study.

Throughout these examinations he is particularly interested to observe that the patient has been faced with and how he has responded or attempted to adjust himself to it, and how he has failed. The general medical man also takes these things into some consideration but, as a rule, without a systematic or persistent attempt to correlate the successive failures or successes of such a patient.

Accordingly, a complete study of the patient, from the psychiatrist's point of view, would also include the analysis of the emotional patterns displayed by the patient in each of the basic situations of life—the home, the school, the vocation, the marriage, and society in general, i.e., his techniques of failing or succeeding with the personnel composing these groups. Finally, a systematic, scientific study of the development of the patient's sexual life cannot be neglected except from hypocrisy or slovenliness.

With all this data (physical-chemical-psychological-social) in hand the psychiatrist asks himself:

(1) What is the insuperable difficulty in the situation which this patient is trying to meet, i.e., what are the extraneous factors impeding normal adjustment? What opportunity does this patient have—now or hereafter—for normal living—physical, mental, sexual, social?

(2) What, on the other hand, is the fundamental weakness or defect in his personality
  (a) From the standpoint of physical structure?
  (b) From the standpoint of physiological or chemical functions?
  (c) From the standpoint of psychological intention and inhibition?

(3) What is the primary purpose of the present illness, and what is the secondary gain accomplished by it?
(4) In an effort to expedite better adjustment of patient and universe,
  (a) To what extent can his environment be changed?
  (b) To what extent can his personality be changed?
  (c) In what way and to what degree may the positive functions of the illness be combated or replaced by more expedient devices?

Once these questions are answered, the psychiatrist feels it is possible to make a diagnosis and outline a treatment in the direction of alleviation, counteraction, correction, and digression. This treatment may utilize not only physical, chemical, and mechanical agencies but psychological and social agencies as well. It will be seen from this how different this is from the technique and the point of view in psychiatry from that of general medicine. It is chiefly a matter of a broader view of the patient and his illness.

The question naturally arises as to whether there is any justification for attempting to bring closer together the viewpoints of the physicians and the psychiatrist. Is it or is it not helpful to consider these instinctual, purposive, emotional factors in general medicine? To the physician they seem unnecessarily elaborate and cumbersome. To the psychiatrist it seems impossible to ignore them with scientific justifiability. Both physicians and psychiatrists are apt to fall back upon the explanation that after all they are dealing with different sorts of patients entirely. But is this really true?

I should not go so far as to say that there is not some pragmatic truth in the distinction that is made between patients who consult psychiatrists and patients who consult general practitioners, but I am certain that these groups are not separated so widely as is ordinarily assumed. The fact of the matter is that the psychiatric approach has never been applied in any systematic or extensive way to the problems confronting the general practitioner. Recently some researches in this direction have been undertaken at the Institute for Psychoanalysis in Chicago and at the Presbyterian Hospital in New York with extraordinary results both investigatory and therapeutic. To speak of the emotional factors precipitating the arthritis, the psychology of colitis, or the psychotherapeutic cure of gastric ulcer would have been regarded as *prima facie* evidence of lunacy or chicanery a few decades ago. Yet all these have been matters of serious scientific consideration by eminent physicians (*not* just psychiatrists) within the past few years.

I should like to suggest a theoretical point of view which might go far toward reconciling these differences of opinion between psychiatrists and general practitioners. This requires a rather radical revision in the ordinarily accepted notions of what disease is and what we, as physicians, are trying to do. Nevertheless, it is an hypothesis which has gradually developed of late on the basis, originally, of empirical observations.

It is generally assumed, I believe, that the sick man comes to a doctor because he has been overtaken by fate, ill-fortune, bacteria, or some other invader—and the disease is looked upon as something the patient hates, fights, and wants to be rid of. He applies to the doctor, who accepts the responsibility and focuses his energies upon combating this foe. But there are many bits of evidence that such a view makes incorrect assumptions and ignores an important principle. It does not require great intuition to detect the fact that the foe with whom many patients fight is not something outside of them but something inside—a part of themselves. They are often ready enough to let the doctors do the fighting for them; some even do their best to oppose his efforts and even though bacteria, bad food, and sharp corners do exist and do inflict wounds, it is often observable that such wounds are invited.

If, in addition to this, we think of the persistence with which some patients cling to their illnesses, the compulsive way in which patients frequently force surgeons to operate and re-operate upon them, the extraordinary repetitiousness of afflictions to which some patients fall victim—these, together with such clinical syndromes as alcohol and morphine addiction, neurotic invalidism, voluntary asceticism from other than religious reasons, malingering, and finally that most dramatic of all human acts, suicide—if we consider these things we must begin to suspect as Freud did that self-preservation is not the only instinct which dominates mankind. On the contrary it would seem as if a self-destructive impulse waged constant battle with the will to live and took advantage of every opportunity to wreak its purpose upon its possessor.

Ordinarily the self-destructive impulses are presumedly held in abeyance. Sick people, on the other hand, may be conceived of as persons in whom the battle has erupted so that they are trying to destroy themselves and at the same time fighting against it, imploring aid in this from the doctor. Such an hypothesis might be applied to

such immediate and sudden self-destruction as is represented by suicide, or to more gradual and diffuse self-destruction such as neurotic invalidism. Perhaps such a thing as tuberculosis in which the individual seems to yield, sometimes all too willingly, to the invasion of an available assailant, and even the more localized or focalized diseases of the body may be thought of as further illustrations. Such an extension of the theory to organic disease was not made definitely by Freud and we are not yet in any position to support it with convincing evidence; it is, however, a logical conclusion from the theory that if there is a self-destructive impulse with the strength which Freud has postulated and which much clinical evidence supports, then we should not be surprised to find that it had an active part in the production of physical as well as psychological disease. Concerning its activity in the latter, (i.e., "mental" disease) we psychiatrists now have no longer any doubt. Concerning its function in the former, we must wait expectantly the result of research.

In terms of this hypothesis, the therapeutic indication is regularly the same, whether in psychiatry, orthopedics, criminology, or cardiology. The physician must throw the weight of his knowledge, his skill, his experience, his chemical, mechanical and psychological adjuvants on the side of the embattled life instinct and in opposition to the destructive tendencies. This is actually what is done at the present time in both medical and psychiatric fields and we know that it sometimes saves the day for the patient. This success, in turn, saves the life of the physician, in a very literal sense. It diverts his own destructive tendencies to the attack on the destructive tendencies of others and by losing our lives, we save them. This probably accounts for the prevalent optimism characterizing physicians in spite of their often dreary experiences and for their conviction that they have this responsibility and this power. Whatever the reasons may be, we physicians well know that our deep feelings and our professional ideals do not permit us to stand by inactively in the face of a life and death struggle. Our daily lives consist in a participation in innumerable minature wars between life and death and our constant striving is to increase our perspicacity, our skill, and our discernment, and, from all of this, our efficacy in the opposing of self-destruction.

And although the phenomenon of war, which now looms again upon the horizon, would seem to be the most dramatic exemplification con-

ceivable of this human wish to destroy one's self (along with one's neighbor) we are simultaneously aware of the weak but growing opposition set up against it by the cooperation of intelligent minorities who struggle with humanity's ills as we physicians wrestle with those of the individual patient.

This is my conception of psychiatry and of general medicine as well, and I feel sure that no one of my colleagues, medical or psychiatric, will deny that from this standpoint, at least, we have more in common than in controversy. What techniques we shall evolve to discover and rout or counteract this self-destruction will differ widely, varying with our talents and our training. But with this concept of disease and medical science we are less likely to be misled by the naive assumptions, false optimism and easy discouragement which the older views of man versus environment inevitably induced.

THE COMPLICATIONS AND SEQUELLAE OF HEAD INJURIES*

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The complications and sequellae of head injuries are of great interest practically to everyone in the medical profession. Their medico-legal significance is sometimes much greater than their medical. I am rather glad that I was asked to speak on the complication of head injuries, as it gives me the opportunity to emphasize what we all know, that it is the injury to the brain and not to the head which is of most importance. If we look through the history of brain injuries practically to within recent times, we find that most of the references deal with the importance of skull fracture. It seems that nobody was interested in the injury to the brain. It is true that in the majority of instances the severity of the brain injury parallels the severity of the injury to the skull; but we also know that there may be a fairly severe blow to the head with no injury either to the brain or the skull and that sometimes a mild blow to the head with no fracture may result in serious brain injury. Sometimes even an explosion at a distance may result in injury to the brain. What I merely want to point out is the fact that the emphasis on fracture of the skull has been altogether too great. Lawyers in litigation cases naturally stress the objective evidence of fracture, no matter what its nature, and often succeed because of this fact in getting damage awards out of proportion to the actual deserts of the plaintiff. On the other hand, many a plaintiff gets less than his deserts, even though there be considerable brain damage, just because there is no objective evidence of fracture.

The sequellae of brain injury may be immediate and remote; the last are sometimes more important. They are characterized essentially by subjective symptoms. But in speaking of sequellae we must make sure that they are definitely related to the head injury. The immediate sequellae are well known and consist of concussion, contusion, and laceration of the brain or hemorrhage within the skull. Their effects usually are apparent immediately or soon after the injury though some

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of the sequellae may not be quite acute or direct. Very often a man receives a blow on the head, loses consciousness temporarily and then has a lucid period. Yet after six, twelve, twenty-four or more hours he may develop gradually hemiplegia. Because of this lucid interval the patient may be neglected and the condition ignored. It is well known that ambulance surgeons sometimes refuse to take a patient to the hospital because by the time they reach the scene of the accident the patient seems to be quite well. It is only the next day that there is gradual onset of hemiplegia and coma and occasionally death supervenes for want of attention and proper treatment. You will recognize in this story the history of middle meningeal artery bleeding. Late apoplexy, stressed mainly by the German school, is a comparatively rare sequel of brain injury. But a man may receive a blow to the head, remain well for a time and then develop a stroke with resultant paralysis. Even though such a patient may have had cerebral arteriosclerosis and develop apoplexy anyway, we may conclude that the blow precipitated the apoplexy which he otherwise might not have had. We must therefore distinguish between precipitation and causation wherever possible. The same question may come up with reference to syphilis of the brain. While paresis must be the result of spirochaetal invasion of the brain, an injury to the head may precipitate paresis or send it galloping onward. It may be that the patient already has paresis and that he may have sustained the fall in the course of momentary loss of consciousness from the disease itself. Similarly an alcoholic may fall during the course of an alcoholic debauch, receive head injuries and then develop a serious train of symptoms. Generally speaking, Korsakoff's psychosis does not follow head injury but acute alcoholic hallucinosis may.

As a rule the sequellae of head injury depend on the intensity of the injury to the brain and its location. Linear fractures are perhaps less likely to be followed by severe sequellae but all fractures may be followed by serious injury to the brain, meninges, and cranial nerves and be characterized by various paralyses. If injury to the cerebrum, more particularly the frontal lobe, is apt to be followed by mental symptoms, injury to the brain and the anterior fossa may result in ocular palsies, blindness and loss of the sense of smell. Deafness and facial palsy are usually the result of injuries at the base of the brain. I think that one thing may be stated with assurance, namely, that with few exceptions

the severity of the brain injury generally runs parallel with the depth and duration of the loss of consciousness. Sometimes unconsciousness lasting twenty-four to forty-eight hours may leave no grave sequellae behind. Generally speaking, the statement holds. From which it follows that the duration of the stay in bed following head injury should parallel the duration of the period of unconsciousness. It is well to keep a person in bed for at least twenty-four hours even after a comparatively short period of unconsciousness.

The question of concussion of the brain is fairly complicated. Occasionally all that this amounts to is momentary "seeing stars." At times a person is merely dazed and unconscious for a few minutes. If the duration of unconsciousness is an hour or two one is justified in saying that the patient has had more than simple concussion. So, too, sequellae are apt to be more severe and longer lasting if the unconsciousness is prolonged. Naturally the sequellae of contusions and lacerations are much more severe. Although not everybody subscribes to this rule, I feel that a lumbar puncture should be performed in every case of brain injury. Not only will the presence or absence of blood in the spinal fluid be determined but the lumbar tap may have considerable medico-legal value. If one can state that lumbar puncture following the injury showed increased spinal fluid pressure and that there was blood in the fluid one can with more certainty prove the existence of brain injury and sustain the patient's claim.

One rather important sequel of head injury which is, I think, becoming more common and which we are becoming more skillful in diagnosing is subdural hematoma. Certainly it is not so rare as we formerly thought. The subdural bleeding need not run parallel with the intensity of the blow. Quite frequently there is a history of only a light or moderate blow to the skull, so slight indeed as to be deemed trifling by the patient at the time of the injury and ignored in the complaint to the physician. The blow may be received in boxing. Generally there is an interval before there is onset of the subdural bleeding. This type of bleeding differs from subarachnoid hemorrhage wherein the blood circulates between the pia and arachnoid, which can be shown on the spinal tap. If the blood accumulates subdurally, external to the arachnoid, the spinal fluid will not show blood. Generally the story is that the patient received a blow, perhaps a minor one, and that he began to suffer from headaches. Weeks or months may pass,

in one instance that I know of more than a year elapsed, before symptoms set in. In any case, there is an interval during which there is little bleeding, cessation of the hemorrhage, then resumption of bleeding. The accumulated blood behaves like a foreign body, really like a tumor. Then the patient shows symptoms of increased intracranial pressure, namely, headache, vomiting, dizziness and possibly choked disks, all depending upon the size and the site of the subdural hematoma. If there is pressure in the motor region contralateral hemiplegia will set in. Sometimes the accumulated blood clots and at others it remains fluid and is encapsulated in a newly formed membrane. Another characteristic is that even after symptoms begin to set in lucid periods may alternate with irrational ones. Ultimately all the symptoms progress and coma sets in. But a patient may even come out of the coma only to have it return. Another characteristic is the presence of ipsilateral signs, that is hemiplegia on the same side as the lesion. One explanation is that the brain is pushed to the opposite side against the skull. The better explanation is the presence of subdural bleeding on both sides. Most surgeons will do a bilateral trephine and turn down a flap on the side where the hematoma is found. On occasion a bilateral craniotomy is necessary.

Besides the numerous kinds of paralyses, from monoplegia to hemiplegia, the patient may be suffering from mental symptoms. An acute psychotic period frequently characterizes many brain injuries. But mental defects may be the permanent results of injury to the brain. These are of particular importance in children in whom they may be very severe. It is noteworthy that children often recover completely from fairly severe brain injuries. On the other hand, damage to a young brain during its course of development is just as likely to stop further development and result in much greater mental defect. Depending upon the age and intensity of the injury, imbecility or idiocy may be the results. An injury to the brain in an old person may leave a greater scar than in a comparatively young one. It is important therefore to take into consideration not only the severity of the injury but the type of brain one is dealing with.

The question of convulsions following brain injury is extremely debatable. Statistics vary to such an extent that it is difficult to evaluate them. In one fairly extensive study of some eighteen thousand soldiers with brain injuries the incidence of convulsions was given

as five per cent. Other observers have given the rate of incidence as high as twenty-two per cent. These differences obviously depend upon the men who compile the statistics and upon the standards of comparison used. Most observers maintain that there should be a definite interval between the date of the injury and the onset of the convulsions. This interval may vary from a few months to many years. Obviously the further one gets away from an injury, say five or ten years, the more difficult is it to correlate the convulsion with the accident. It should be emphasized that convulsions may occur during the course of an acute period following brain injury. Here we are not dealing with so-called traumatic epilepsy. At times it is possible to correlate convulsion with the actual site of the injury. Focal convulsions may result from direct injury to the motor region. Such convulsions may be due to depressed fractures or scars. Generalized convulsions may occur from injury to any part of the brain, although injuries to the anterior part of the brain are more likely to be followed by convulsions. Slight injury may give rise to scars which will ultimately eventuate in general or focal convulsions. If one is fortunate in localizing the scar and excising it one may put a stop to the convulsions. The important point is that one cannot predict from any given injury whether a person will develop convulsions and how soon.

I come now to what I regard as a very important subject, namely, the post-traumatic syndrome. There is considerable confusion regarding the subject, all the more as we are dealing with intangibles and largely subjective disturbances. If a patient says that he has a headache he expresses a subjective sensation and you may not be able to verify it objectively, although severe pain may show itself in facial expression. Similarly if a patient says he is dizzy one is again confronted with a subjective complaint. We can evaluate vertigo objectively by means of nystagmus, falling, nausea and vomiting but subjective vertigo must be taken at the patient's word. So, too, when a patient says, "I do not remember" it is difficult to say whether or not he has true memory defect. The emotional disturbances such as irritability, lability of mood, tendency to weep, anxieties are also very difficult to evaluate. Fatigability and insomnia belong in this group. We have, then, a congeries of symptoms which have received various designations. Thus, we speak of these syndromes as concussion, traumatic neurosis, traumatic hysteria, shell-shock, neurocirculatory asthenia, neurasthenic

syndrome, nervous exhaustion and post-traumatic state. They characterize apparently one group of symptoms. If we could come to some better understanding we could at least speak a common language and not contradict each other when we come on the witness stand.

It seems to me that the whole post-traumatic syndrome can be divided into several categories. The first may be called litigation neurosis, or better still, malingering. A man may feign all symptoms, including headache and dizziness and poverty of memory, and may make it very difficult for us to detect the truth. A person may certainly make a tremor realistic and occasionally also a convulsion. In those cases we are dealing with individuals who, for the purpose of gain or the evasion of responsibility, allege the presence of symptoms which they may have had but certainly do not have at the time of the complaint. In these cases we are dealing with conscious, purposive deception. Fortunately this group is comparatively small. More common is the general tendency to the exaggeration of symptoms. Most of us are prone to exaggerate complaints if a motive enters into it. This is particularly true if a kind physician or interested lawyer helps the patient to exaggerate the injury. Occasionally there is an "incubation period" between the date of the accident and the onset of the alleged symptoms. As a matter of fact malingering is more common among psychotics than neurotics. In any case, it is frequently difficult to be absolutely certain in one's opinion.

As to concussion, it is fair to say that we are gradually getting away from the old conception. Indeed there are those who would discard the word and substitute the term encephalopathy. In any case, the words molecular disturbance are quite meaningless. In true concussion or encephalopathy there are minute hemorrhages, disturbances in the circulation of the cerebral spinal fluid, and ultimately distortion of the ventricular system, atrophy of the brain and perhaps adhesions in the meninges. Martland and others have pointed out these facts and particularly called attention to what happens in prize fighters. It is no secret that a famous prize fighter gave up his career because of the advent of headaches which portended serious brain injury. Very frequently one observes the organic changes on encephalographic studies. Such brain injuries are particularly common among prize fighters. Sometimes the boxer is knocked out, as it were, on his feet and keeps on fighting. We praise his gameness at a time when the man is prac-

tically unconscious and we do not know that he keeps fighting automatically. Parkinson syndromes may develop in prize fighters because of repeated minute hemorrhages. Occasionally encephalitic syndromes not due to infection may result from repeated head injuries. Very frequently following concussion encephalographic studies show nothing and yet six months or a year later definite evidence may be produced. It is quite possible that many patients whom we call neurotic and hysterical are not so in a psychogenic sense but have actual brain injury. This does not exclude the possibility of additional psychologic factors.

Traumatic hysteria is perhaps the most common sequel of trauma. If our conception of hysteria is correct, trauma alone cannot cause hysteria. Certainly from the point of view of psychoanalysis hysteria must be traced back to childhood patterns. That is to say, we are dealing with a personality which is conditioned to the development of hysterical manifestations, a personality which is already in conflict with itself. Trauma becomes a focal point around which the symptomatology is built, so that you may say that trauma precipitates the hysterical condition but does not truly cause it. To make the diagnosis positive, however, one must show that there was an injury; that following the accident, perhaps after the incubation period, the individual developed the hysterical syndrome; but we must insist that there are unconscious factors in addition to the trauma. The question arises why should a laborer who earns, say fifty dollars a week, wish to give it up for a compensation of perhaps ten or fifteen dollars. It may be that he has had conflicts over his work before the accident. The trauma acts as a release, as an outlet. It is necessary, therefore, to make personality studies in such cases.

The prognosis obviously depends upon many factors, most of which are personal ones. If one can remove the litigation motive there is a much better chance of treating the hysteria. That is why it is so important to settle such cases as quickly as possible. If it is not immediately settled one may complicate a situation which is already sufficiently complex in itself. My advice to lawyers and insurance companies invariably is to settle cases as quickly as possible. In medicolegal complaints in New York City it takes two to three years before a case comes to trial. The fact is that most cases can be properly evaluated within a reasonable time after the accident; anyway, one can always reopen a case.

There is a fourth group which presents practically the same symptomatology in which there is apparently no encephalopathy, no malingering and no hysteria. An iron worker, for instance, may fall off a beam and receive a blow to the head. The injury may be slight and the period of unconsciousness short, even though there was at the time of the fall a grave threat to life. The symptomatology in such cases sometimes simulates those syndromes seen during the war when brave soldiers and officers who received slight injuries could not return to the front. Many of those patients had frightful nightmares in which they relived their harrowing experiences. So, too, the iron worker may be unable to return to his occupation. The question that I raise is whether we may not speak of such cases as traumatic neuroses, giving the neurosis a much deeper significance. Perhaps we are dealing with an ego neurosis wherein there was not a threat to the libido but to the ego, to the life instincts. Such patients are not amenable to psychotherapy, such as hypnosis or analysis, nor does medication help. Fortunately we are dealing here with a comparatively small group.

The submission of these four syndromes obviously is tentative. The important point is for us to agree on the terms so that we may better understand each other and use them until such time as we can substitute better concepts. It really does not matter for the moment whether we are entirely right or not, but we must have working definitions. In any case, I submit these concepts for your consideration and discussion.

DISCUSSION

Dr. Roback: What is the relation of trauma to brain tumor?

Dr. Wechsler: Doctors Kernohan and Moersch have done some work on this subject and practically proved that gliomas never follow head injuries. I think that certain types of tumors may be correlated with trauma. Sarcoma may follow trauma to the skull as it does elsewhere in the body. Endothelioma is said in rare cases to follow trauma; I doubt it. Ninety-nine out of one hundred cases have no history of trauma, however. A man may have a tumor and fall and strike his head and develop a train of symptoms which belong to the tumor. There must be an interval of at least three months between the injury and the onset of the symptoms. A meningioma may exist for more than a year before symptoms occur, and so may gliomas. Some tumors can infiltrate a tremendous area of the brain without giving symptoms. I think we can safely exclude all tumors of the gliomatous group; and I doubt whether meningiomas can follow injury. Sarcomas can. In any case, the influence of trauma is overemphasized.

Dr. Reider: How frequently would you say there is evidence of encephalopathy with out and out hysterical manifestations? What can be done for these?

Dr. Wechsler: I once saw a lawyer, a man who was apparently very well adjusted, who was hit by an auto. He had telescopic vision, hemihypalgesia, and other out and out hysterical symptoms. An encephalogram was normal. He continued over a period of three years in this way. As he didn't get well we did another encephalogram. This time it showed dilated ventricles, etc. Here we had the mixture of hysterical manifestations with definite evidence of brain damage.

I think I mentioned that there are generally likely to be psychological factors and that even the organic manifestations are colored by the personality of the individual. It depends upon the skill of the examiner to dissociate the two. How much hysteria is due to actual brain injury and how much brain injury is initiated by psychological factors is difficult to determine. This is a very important question. Both are possible. Unrelieved fear may initiate a goitre and cause actual hyperthyroidism. In traumatic hysteria the mechanism is unconscious; you must accept the theory of the unconscious and I think you must say that the symptoms of a traumatic hysteria are unconsciously determined.

Dr. Stone: What is the therapeutic benefit of encephalography?

Dr. Wechsler: The theory is that air breaks up some adhesions in the subarachnoid space. Some believe that this air changes the intraventricular dynamics and initiates recovery. Whether this is true or not I do not know; the fact is that in some cases encephalography does have the therapeutic effect.

Question: Do you think the depth of the unconsciousness is more important than its duration?

Dr. Wechsler: The duration is perhaps more important than the depth. If a patient has lost his corneal reflexes he is apt to be unconscious for a long time. The period of coma may be brief even in very severe injuries. Unconsciousness does not always mean coma. A patient may be delirious and at the same time be unconscious of what is going on. He may have complete amnesia for the period and not be in coma. Generally there is a definite correlation between the depth of the unconsciousness and its duration.

ACUTE HEMORRHAGIC ENCEPHALITIS (PERICAPILLARY ENCEPHALORRHAGIA) FOLLOWING ARSPHENAMINE ADMINISTRATION*

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Introduction. Among the severe although relatively infrequent complications of treatment with arsphenamine or neoarsphenamine is acute hemorrhagic encephalitis. In the literature a multiplicity of terms has been used to describe this condition, such as brain purpura (Schmidt), serous apoplexy (Netherton) (5), medullary perivascular necroses (Alpers) (3), toxic encephalopathy, and finally pericapillary encephalorrhagia (Globus and Ginsburg) (4). The term "hemorrhagic encephalitis" persists and is here employed as a purely clinical designation of the condition.

Glaser, Imerman, and Imerman (1), in their statistics collected from several sources, state that 191,125 patients receiving 1,108,778 injections of arsphenamine compounds had 1,377 complications. One complication occurred in every 129 cases and in every 804 injections. One death occurred in every 2,699 cases and in every 14,384 injections. Of the total number of deaths about 50 per cent were due to central nervous system lesions. Thus, approximately one death was due to central nervous system lesions in every 5,398 cases and in every 28,776 injections. Of the remaining 50 per cent of deaths, most were caused by exfoliative dermatitis and acute yellow atrophy of the liver; in rare instances, by acute hemorrhagic nephritis, ulcerative enteritis, and aplastic anemia.

Arsphenamine encephalitis has already been subjected to extensive

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clinical and pathological study (1, 2, 3, 4, 6, 7, 8, 9). However, the following case is presented as a strikingly clear example of the clinical syndrome and the pathologic anatomy.

History. May 3, 1935, a housewife, 32 years old, was admitted to Christ's Hospital, Topeka, Kansas, complaining of aching all over her body. She was extremely tense and irritable. She had always had satisfactory health except for a vague story of muscular spasm occasionally occurring at night. This was said to have involved various parts of her body, most frequently the right arm. About six weeks or two months before admission, without any apparent cause, she developed an acute cystitis characterized by painful urination. She was treated medically, and because she could not tolerate oral medications, she was given a few small injections of neosalvarsan, about three doses in all. The patient improved considerably, but a few weeks later she had a return of symptoms and was given another injection (0.45 gram) on April 14, 1935. It was said that the patient "reacted badly" to each of these injections. On May 2, 1935, she began to vomit profusely and continuously, and when her condition did not improve with symptomatic therapy, she was admitted to the hospital for more intensive study. A small abscess in the right axilla had been "recently" incised; the date was not certain.

Examination. The physical examination revealed an emaciated adult female, lying in bed, extremely tense and complaining of aching all over. A small right axillary scar was present. Nothing abnormal was discovered in the heart, lungs, or abdominal organs. Blood pressure was 126/80, pulse 92, respiration 22, and temperature 97°F. (R). The extremities were normal, but deep reflexes were exaggerated and equal.

On May 4, 1935, the patient passed into coma and the temperature rose to 105°F. (R), pulse 108, and respiration 28. At this time a neurological consultation was requested.

Neurological examination. Breathing was regular and somewhat rapid. The patient responded very slightly to prolonged painful stimuli. The head was drawn to the right and slightly backward. There was some uniformly increased plastic tonus of the neck, not typical "meningeal" rigidity. The right arm was in flexion spasm across the chest, all of the fingers flexed except the middle finger, which was extended; the left arm was in extension spasm except the hand, which was flexed. The left leg was slightly flexed, and the right leg extended. Both feet were in a partial "equinovarus" position. The eyes usually deviated very slightly to the right, but roved occasionally. The pupils were round, regular, equal, and myotic, reacting with very limited excursion to light. Corneal reflexes were bilaterally present, but diminished. The fundi were normal. There was no gross facial

weakness. The reflexes of the lower extremities were symmetrically hyperactive; there was no ankle clonus. The legs were flaccid. No definite Babinski or other pyramidal tract sign was present. The reflexes in the right upper extremity were hyperactive; those in the left upper extremity were within normal limits. The abdominal reflexes could not be obtained. The right arm was spastic on attempted extension, and the left arm spastic on attempted flexion. Detailed sensory examination was impossible. The patient was not incontinent of feces. There was retention of urine, requiring catheterization.

Two spinal punctures were performed, one on May 3, 1935 (before consultation) and the other on May 4, 1935. The first spinal fluid was grossly (uniformly) bloody, but the second performed May 4, 1935 was quite clear after a few cubic centimeters of blood-tinged fluid were removed. Manometric determination of pressure was not carried out.

Laboratory examination:

Urine: (catheterized specimen) (5/4/35) straw, slightly cloudy. Specific gravity 1.028. Albumin negative; sugar three plus (qualitative); acetone one plus; diacetic acid two plus. Microscopic: 30–40 pus cells per high power field.

Blood: (5/3/35) Erythrocytes 5,670,000, haemoglobin 87 per cent, leucocytes 5,800, polymorphonuclears 76 per cent, small lymphocytes 24 per cent; (5/4/35) erythrocytes 4,960,000, haemoglobin 85 per cent, leucocytes 16,450, polymorphonuclear 95 per cent, small lymphocytes 5 per cent.

Fasting blood sugar: 166 mgm. per 100 cc. of blood (before intravenous glucose).

Blood Wassermann and Kahn: Negative.

Spinal fluid: (5/4/35) Red blood cells 50 per cubic millimeter, white blood cells 5 per cubic millimeter, total protein 420 mgm., globulin two plus, Wassermann and Kahn negative, colloidal gold curve 0000011000.

The working clinical diagnosis was *acute hemorrhagic encephalitis probably due to arsphenamine*. However, in view of the peculiar history of spasms of the right arm, the possibility of a focal lesion in the left cerebral hemisphere, possibly a cerebral aneurism which caused some seeping of blood and finally a frank subarachnoid hemorrhage, was given some consideration. In view of the history of an unexplained cystitis in a young adult, the possibility of tuberculoma with atypical tuberculous meningo-encephalitis was also taken into account.

Course in hospital. On May 5, 1935, the patient developed acute pulmonary edema and was slightly cyanotic. Temperature was 103.2 (R), pulse 160, and respiration 44. The patient was treated very energetically with atropine, glucose 50 per cent intravenously, sodium thiosulphate 10 per cent intravenously, and frequent doses of adrenalin (1:1000). Since she had hyperglycemia and glycosuria, she was given insulin. Neurological examination showed: Rapid gasping respira-

tion, almost entirely abdominal in type. The right side of the face was smoothed out, and there was a bilateral Babinski sign. There was no rigidity of the neck.

May 6, 1935. There was some improvement. No Babinski could be elicited. Temperature was 104° (R), pulse 152, and respiration 24. There was no change in body posturing.

May 7, 1935. Temperature 102° (R), pulse 104, and respiration 24. The pupils which had responded to light to date were absolutely fixed. The patient died about 10:30 A.M.

Postmortem examination. Autopsy was limited to the head. The brain was extremely edematous and the pial vessels were very much congested. The convolutions were moderately flattened out and the sulci appeared shallow. On preliminary section, the brain showed massive petechial hemorrhages throughout the whole of the cerebral white matter. After hardening the brain in 10 per cent formaldehyde, it was sectioned frontally (fig. 1).

In the left frontal pole there was an area of grayish softening about 2 by 3 cm., with marked intrinsic and circumjacent petechial hemorrhages. There were massive hemorrhages throughout the corona radiata, predominantly subcortical. There were many petechial hemorrhages in the corpus callosum. In the anterior portion of the mesencephalon masses of hemorrhages confined to the cerebral peduncles were seen. More posteriorly there were hemorrhages in the red nucleus, and still further caudad, there were hemorrhages immediately ventral to the aqueduct of Sylvius. As shown in figure 1, there were massed petechial hemorrhages in the pons and in the cerebellar peduncles. There were no hemorrhages in the cerebellum. In the medulla oblongata, punctate hemorrhages could be observed in the pyramids. There were a few hemorrhages in the hypothalamic region (related to the hyperglycemia?) and some subpial hemorrhages over the cortex.

Microscopically the lesions appeared as "ring hemorrhages" (fig. 2). Under high power (fig. 3), in the center of each hemorrhage there was a precapillary filled with eosin staining hyalin-like thrombus. The endothelial cells of the central precapillaries were swollen and quite distinct. Around the central precapillaries there was a zone of glia cells, free from hemorrhage, and around the zone of glia cells, there was a ring of massed red blood cells in an excellent state of preservation. There was no evidence of any acute or chronic leucocytic inflammation.

A detailed histologic study of the tissue was carried out in the neuropathologic laboratory of Mount Sinai Hospital of New York.

Microscopic anatomy. The meninges. They were slightly thickened. In places the arachnoid membrane was thickened with cells heaped up to form plaques. The pial vessels were dilated and filled with blood. There was an occasional small subpial hemorrhage and an accumulation of small round cells in the pia.

The cortex. Only a few ganglion cells demonstrated pathologic changes. Where such changes were present they consisted of pyknosis

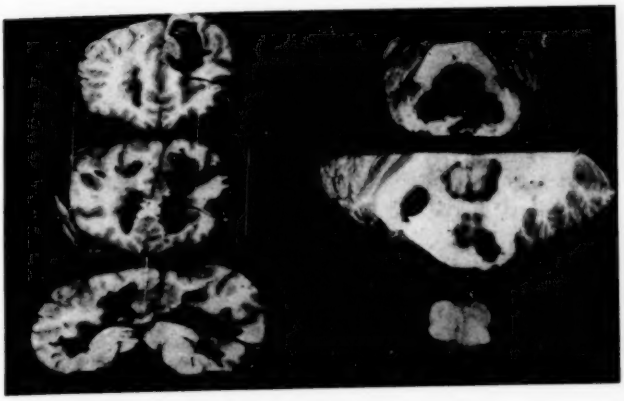

FIG. 1. GROSS SPECIMEN SECTIONED FRONTALLY, SHOWING EXTENSIVE HEMORRHAGIC LESIONS, CONFINED TO THE WHITE MATTER, EXCEPT IN MIDBRAIN

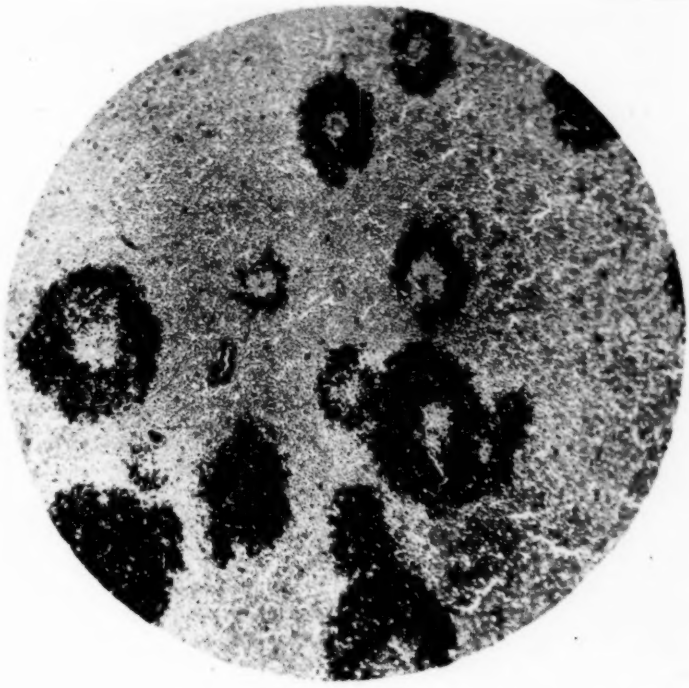

FIG. 2. LOW POWER MAGNIFICATION OF LESIONS, SHOWING "RING" STRUCTURE. HEMATOXYLIN-EOSIN

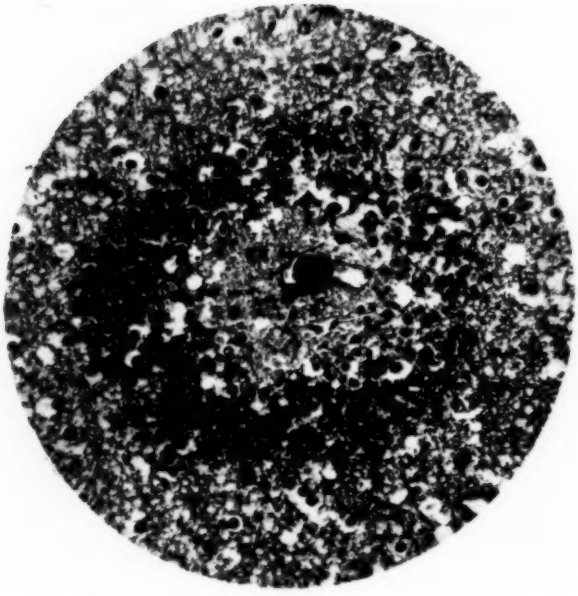

Fig. 3. High Power Magnification of Lesion, Showing Intact Central Precapillary with Circumjacent Hemorrhages. Hematoxylin-Eosin

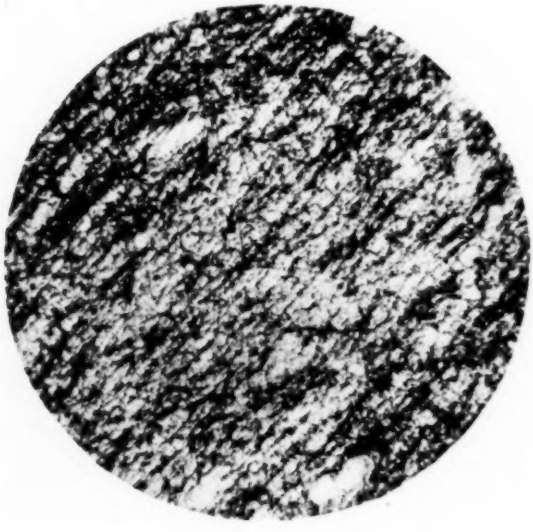

Fig. 4. Myelin Sheath Stain, Showing Changes in Myelin Sheaths in a Hemorrhagic Focus

of nuclei and mild attenuation of cell processes. The Cajal gold sublimate stain (Globus Modification) demonstrated an increase in astrocytes with an occasionally interspersed cell of Hortega. No hemorrhages were found in the gray matter. Slight congestion of vessels and swelling of the endothelial lining of some vessels were the only vascular changes noted.

The white matter. The subcortex throughout its depth was punctuated with hemorrhages. These extravasations of blood assumed a ring form with a clear central area. Hemorrhagic foci of an apparently globular character, without central clear areas, were also noted. There was a marked gliosis and distinct evidence of mobilization of glia about blood vessels.

The medulla oblongata. Here only occasional ring hemorrhages were found in the long fiber tracts.

The vessels in the subcortex showed, in areas, moderate congestion, but deep within the white matter distinct pathological alterations in the vessel walls were found. Many of the lumina of vessels seemed collapsed. The endothelial lining was swollen, the individual cells distorted and taking stain poorly. In some vessels there was desquamation of the endothelial lining to such an extent that the lumen seemed filled with leucocytes (leucocytic infarct). An occasional vessel (same area) showed perivascular infiltration with large endothelioid cells and, in areas, dilatation of the perivascular spaces suggestive of edema. One small area showed disorganization of the stroma about a vessel in which were found leucocytes and large round cells with vacuolated cytoplasm (*gitter cells*). Certain of the larger vessels showed hyalinized material about them.

A thorough study of the "ring hemorrhages" failed to demonstrate a ruptured vessel. The red blood cells in the ring were intact. In many instances there was a central vessel which contained red blood cells within its walls. Occasionally, tiny capillary branches of these central vessels (or precapillaries) radiated out toward the "ring hemorrhage." Red blood cells were also found within these capillaries. The so-called central free zones were not entirely devoid of disease alterations. A fibrin-like network surrounded the central vessel and also radiated for short distances from around the "ring hemorrhage."

The myelin sheaths appeared normal except where there were hemorrhagic foci. Here they appeared swollen, twisted, and their quantity was considerably thinned out (fig. 4). The myelin sheaths distal to the hemorrhage also appeared somewhat thin. Fat globules were demonstrated about a number of the vessels but were nowhere found in large numbers. The astrocytes were large, tended to be fibrous, and were present in great numbers. The axis cylinders seemed to course through the hemorrhagic areas with but slight interruption.

On further study the lesion in the left frontal lobe, which seemed grossly dissimilar to the other lesions, was found to exhibit the same histopathology.

Clinical comment. In this case, the interval between the last dose of arsphenamine and the onset of encephalitic symptoms was unusually long. However, even longer intervals have been reported. The usual interval is from 12 to 144 hours after injection (1). In this instance, there is no certainty that the patient received no arsphenamine between the last known injection (i.e., the one given by her regular physician) and the onset of symptoms.

It is known that the patient was suffering from a genitourinary infection, and since hemorrhagic encephalitis can occur in the course of other infections as well as in the form of a primary (probably virus) disease, it is possible that the presence of infection contributed a toxemia favorable to the development of encephalitis. There was no evidence of metastatic brain infection. It is significant that the patient did not have syphilis, which, of course, is the principal indication for the use of arsphenamine. This strengthens the theory that syphilis per se has nothing to do with arsphenamine hemorrhagic encephalitis (1, 4).

The development of symptoms in this patient occurred after only "a few" injections. This is the usual observation (1). The fact that the patient reacted adversely to each injection might indicate that such reactions should always be taken very seriously with regard to the possibility of a grave complication.

While some reasonable scientific doubt (on a clinical basis) might persist regarding the direct connection of the encephalitis in this case with the arsphenamine therapy, the presence of a highly characteristic tissue reaction makes the connection almost certain.

Discussion of pathology. "Arsenic in organic combination exerts in a very small number of cases a specific action on the capillaries of the white matter of the brain and spinal cord that leads to hemorrhage (6)." The condition in which such miliary hemorrhages occur following administration of arsenical organic compounds has frequently been referred to as "acute hemorrhagic encephalitis." The hemorrhages are often limited to the centrum semiovale, corpus callosum, and internal capsule. They occur infrequently in the gray matter of the brain, but when found in the gray matter they are most abundant in the basal ganglia, especially in the thalamus, and occasionally in the caudate and lenticular nuclei. On histologic examination the lesion consists of a central capillary or precapillary vessel with swollen or destroyed

endothelium. Usually the lumen contains a hyaline thrombus. Immediately next to the vessel is an area of necrosis, completely surrounded by a palisade-like arrangement of glial cells (Spielmeyer). Beyond the collar of glial cells, which (according to Alpers) are oligodendroglia, is a wide ring of red blood cells. There is no evidence of any acute or chronic leucocytic reaction. For this reason, Globus and Ginsburg call this condition "pericapillary encephalorrhagia." The ring hemorrhages according to the latter authors are due to the rupture of tiny capillaries which surround the precapillary vessels. They take the form of a partial or complete ring depending on the extent of capillary destruction. Ring hemorrhages are encountered in other conditions of the brain; for instance, in the vicinity of brain tumors and inflammatory lesions. Ring hemorrhages are also seen adjacent to more massive solid extravasations. But it is the predominance of such hemorrhages that makes this lesion characteristic of arsphenamine encephalitis.

There is some difference of opinion regarding the exact structure of the foci in the central nervous system. Schmidt (quoted by Alpers) (3) believed that the origin of the focus is a perivascular hemorrhage which is the result of diapedesis of red blood cells following injury to the vessel. He was sufficiently impressed with the presence of erythrocytes in these areas to give the term "hemorrhagic encephalitis" to this condition. On the other hand Spielmeyer thought that the foci are independent of the hemorrhages and that the important factor is a central necrosis as a result of injury to the vessels either by toxin or by infection. Alpers, agreeing with Spielmeyer, emphasized the perivascular necrosis and suggested the term "medullary perivascular necroses." In our case, the feature of pericapillary hemorrhage obviously predominates, and since there is no conclusive histologic evidence of inflammatory reaction, the term proposed by Globus and Ginsburg is probably best suited to the pathologic anatomy.

Conclusions. A case of hemorrhagic encephalitis (pericapillary encephalorrhagia) following the treatment of a urinary infection with neoarsphenamine has been presented and discussed from the clinical and pathological points of view. It is felt that while a grave complication of this type is infrequent in arsphenamine therapy in general, its extreme malignancy may well raise the question of the advisability of special precautions in the use of arsphenamine or its derivatives in the

treatment of urinary infections, where two special hazards are implicit in the disease being treated: (*a*) the presence of infection with toxemia; (*b*) the presence or imminence of some degree of renal embarrassment.

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PSYCHOTIC REACTION IN CARBON MONOXIDE POISONING

By William C. Menninger, M.D.

Psychosis due to carbon monoxide poisoning is rare as judged by the remarkable figures reported by Shillito et al. (1) covering the ten year period from 1925 to 1935 of 21,000 instances of carbon monoxide poisoning which were responded to by emergency squads in the New York metropolitan area. Approximately one-third of the patients died. These investigators combed the state hospital records serving this area and found that there were only 39 cases of psychosis due to carbon monoxide poisoning out of 81,659 admissions.

In a series of 43 cases of carbon monoxide poisoning seen while in coma I have reported the pupillary findings (2). Eight of these patients died. So far as is known none of the others developed sequelae though satisfactory follow-up studies after discharge were not possible. The case here presented represents a severe psychosis associated with carbon monoxide poisoning and has three special features. 1. The individual apparently was in a state of agitated depression at the time he was poisoned and this undoubtedly colors the symptomatic picture of the psychosis. 2. He presented a peripheral neuritis; this is a frequent complication of carbon monoxide poisoning as reported by Grinker (3), Wilson and Winkleman (4), Solomon (5), and Mankowsky (6). 3. He has shown a very slow improvement and will probably recover.

The patient was a 35-year-old lawyer, the oldest of a family of five children born to healthy parents. He finished his pre-law work with some difficulty, since it was necessary for him to work, and graduated in law from an eastern university with a good scholastic record. During his first year of apprenticeship he became involved with a girl who became pregnant; he developed a depression which lasted several months. Three years later, at the age of 28, he developed a second minor depression, again following a disappointment in love. At the age of 31 he married, though was unhappy with his wife over minor questions of morality. Two years after the marriage she died in child-

birth though the child lived. The patient employed a housekeeper to take care of the child, and over the next two years became involved with her. He became anxious, fearful, and depressed; he believed that he was being photographed in his relations with this girl, and that someone was going to get his job.

He consulted a psychiatrist on January 8, 1935 and again on January 26th; both times he was markedly agitated, and reported essentially the same story. On February 6th he was found unconscious in his car in his garage, with the ignition turned on but the motor not running. He was taken to a general hospital where he was lethargic for four days; after that he was unable to remember faces or names or any recent events for more than a few minutes. Examinations of his spinal fluid, blood, and urine all were normal, though no satisfactory test was made for carbon monoxide at that time. Because of his mental condition he was admitted to the Menninger Sanitarium on February 19th.

At that time he showed no physical abnormalities, but neurological examination showed a neuritis of the right posterior femoral cutaneous nerve with marked motor involvement. Laboratory examinations again were negative, however, except that the spectroscopic test of his blood showed definite bands in the spectra limited to the carbon monoxide area. His mental picture showed a complete amnesia for the preceding eight weeks, except for a few isolated events which he vaguely remembered. He did not recall consulting the psychiatrist and had no memory of any anxiety or concern about his affairs. He was unable to remember his attending physician for an interval of ten minutes, or an incident which occurred only an hour previously. He was depressed and cried frequently. His remote memory seemed reliable. He showed no calculation defect and no other intellectual defect. He was unable to show any interest in those about him.

The patient was observed over a period of ten days and at that time discharged to his family. A report thirteen months later from a physician who has taken care of him stated that he was still depressed, had little interest in things, and still had some amnesia, though he had shown marked general improvement.

The diagnosis in this case seemed very definitely established from the history of probable exposure (which may or may not have been a suicidal attempt), the spectroscopic test, the presence of a marked amnesia, and the peripheral neuritis. It is important to recognize,

however, that the mental picture undoubtedly combined the direct effects of the carbon monoxide poisoning and the agitated depression. It is now a widely accepted fact that in every organic or toxic psychosis the mental picture represents the pharmacological effect of the poison superimposed upon the individual personality make-up. Among others, Alfred Gordon (7) called attention to the psychiatric significance of the special symptoms resulting from carbon monoxide poisoning. In the case reported herein the particular form of the patient's mental symptoms served to blot out the immediate cause for his great agitation, and permitted him to regress to a childish level at which someone would necessarily look after him.

The onset of the symptoms in this reported case was immediate. In 19 of the 43 cases collected by Shillito et al. (1) the onset of symptoms occurred within the first week, the majority of patients going directly from their unconscious state into a confused and disoriented state. It is of interest to note that 13 of their cases presented a week or more of mental clarity following the acute poisoning; the longest period of clarity was 19 days.

The mental symptoms which this patient showed, i.e., depression and amnesia without hallucinations or delusions, are typical of the psychotic reaction in this condition. Despite the contribution of the prepsychotic personality defects, the psychotic reaction in carbon monoxide poisoning is remarkably stereotyped. As a rule, the individual awakes from his coma in a confused and disoriented state with no recollection of recent events. The confusion may last for weeks though it is rare that the patient becomes either hallucinated or delusional. He may become oblivious to his surroundings with a marked psychic regression. Emotionally he is usually dull and apathetic, and occasionally depressed. In the recovery, the confusion clears before the amnesia, the latter being the most frequent mental symptom in this condition.

Neurological signs are frequently present in carbon monoxide poisoning. The most common of these is hypertonia of the peripheral reflexes. In this case the patient developed a true organic neuritis of the right lower extremity, probably accompanied by some sacral radiculitis. He was very sluggish in his movements and had a slight tendency to a fixed facies though did not show any definite evidence of Parkinsonism as reported in cases by Grinker (3) and Mackay (8). The neuritis

showed a very marked improvement during the ten days he was under observation.

As to the outcome in carbon monoxide psychoses, Shillito (1) reported that 23 out of 43 cases recovered, 11 died, and 9 suffered permanent effects. The 11 patients died within two years after the acute poisoning; the 23 patients were paroled or discharged without any apparent permanent scars of the poisoning.

The pathology of this condition has been repeatedly studied and is excellently summarized by Grinker (3). The initial vascular paralysis is followed by asphyxia of structures having a poor capillary supply. In many instances there seems to be an acute lenticular degeneration, but there is fairly uniform agreement that the mental symptoms are due to diffuse cerebral lesions and are not pallidal in origin. Mankowsky (6) believes that the peripheral neuritic symptoms result from a collapse of the nerves by hemorrhage which originates from the fragility of the vessel, and from the infiltration of the epineurium and the perineurium with blood. The result is scar formation and ischemia of the nerve.

SUMMARY

A case of carbon monoxide poisoning followed by a psychosis is presented; the patient showed a peripheral neuritis, the mental symptoms of depression and amnesia, and had shown marked general improvement when last heard from some 13 months after poisoning.

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BOOK REVIEWS

Practical Aspects of Psychoanalysis. By LAWRENCE KUBIE, M.D. Price, $2. Pp. 223, New York: W. W. Norton, 1936.

In general, this is an authoritative and valuable book, especially for the medical profession. However, some of the author's dogmatic statements regarding the necessity for frequency of analytic sessions, absolute analytic incognito, and artificial deprivation have antagonized many of his colleagues. A serious omission is the lack of a discussion of analysis in hospitalized cases. (R. P. K.)

The Principles and Practice of Recreational Therapy for the Mentally Ill. By JOHN E. DAVIS in collaboration with WILLIAM R. DUNTON, JR., M.D. Price, $3. Pp. 206. New York: A. S. Barnes, 1936.

This book gives the impression that the author has had a great deal of experience in recreational therapy, as it records many excellently presented observations, particularly with regard to swimming. The author's psychiatric attitude is limited chiefly to the symptomatic approach, and his classification and psychiatric concepts and terminology are distinctly descriptive. At no place does he consider unconscious conflicts or psychic dynamics. (W. C. M.)

The Nature of Human Conflicts. By A. R. LURIA. Translated from the Russian by W. Horsley Gantt. Price $4. Pp. 431. New York: Liveright, Inc., 1932.

Luria describes the technique of his experimental procedures and explains the various meanings of the results of some of the experimental findings. One of his assumptions is that the left-hand response is evidence of affective disturbance in connection with the stimulus, and he concludes that this affect response has its origin somewhere in the previous life of the individual in relation to some conflict. (N. R.)

A Study of Masturbation and The Psychosexual Life. By JOHN F. W. MEAGHER, M.D., F.A.C.P. Third edition. Revised and re-edited by Smith Ely Jelliffe, M.D., Ph.D. Price $2. Pp. 144. Baltimore: William Wood & Co., 1936.

This is a very good compendium written in relatively simple language, discussing, in general, psychosexual life, with about two-thirds of the book devoted to a discussion of masturbation—its causes, the reactions to it, symptoms, and finally a rather simple but helpful discussion of the treatment. (W. C. M.)

RECENT PUBLICATIONS BY MEMBERS OF THE STAFF

MENNINGER, K. A.: *Purposive Accidents as an Expression of Self-Destructive Tendencies*, Internat'l. J. Psa. **17**: 6–16, Jan. 1936. (Also in German in the Almanach der Psychoanalyse. Vienna, 1936.)

The paper presents the thesis that accidents are often purposeful to fulfill an unconscious intention of self-destruction. These motives include elements of aggression, punition, and propitiation with death as the occasional but exceptional outcome. Numerous examples of purposive accidents are given.

MENNINGER, K. A. AND MENNINGER, W. C.: *Psychoanalytic Observations in Cardiac Disorders*. Am. Heart J. **11**: 10–21, Jan. 1936.

A review of the literature of the psychologic factors in cardiac disorders is concluded with an intensive psychologic study of three cases. An attempt was made to show that the emotional factors associated with disorders of the heart are capable of more explicit designation; in the cases studied, it would appear that heart disease and heart symptoms were a reflection of strongly aggressive tendencies which have been totally repressed.

KEMP, J. E. AND MENNINGER, W. C.: *The Influence of Inadequate Treatment of Early Syphilis on the Incidence and Incubation Period of Neurosyphilis*, Bull. Johns Hopkins Hosp. **58**: 24–34, January 1936.

From a study of 680 patients with complete data, 265 of whom had neurosyphilis, it was found that inadequate treatment given within two years after infection did not increase the incidence of neurosyphilis, and in females it appeared that such treatment lowered the incidence of neurosyphilis. Early inadequate treatment reduced he incubation period of neurosyphilis approximately five years.

MENNINGER, W. C.: *Individualization in the Prescriptions for Nursing Care of the Psychiatric Patient*, J. A. M. A. **106**: 756–761, Mar. 7, 1936.

This paper outlines the methods used to train psychiatric hospital personnel, the prescription of a program of treatment for the psychiatric patient, and the methods used by this personnel in carrying out this program. A detailed presentation and discussion of the physician's orders is included, illustrated in its application by a case presentation.